ENCLOSURE 6

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HON. JOHN C. COUGHENOUR

IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF WASHINGTON

DIOXIN/ORGANOCHLORINE CENTER, and COLUMBIA RIVER UNITED,

Plaintiffs,

v.

DANA A. RASMUSSEN, et al.,

Defendants.

No. C91-1442-C

DECLARATION OF DONALD C. MALINS

I, DONALD C. MALINS, declare as follows:

1. My name is Donald C. Malins. I hold Doctor of Philosophy and Doctor of Science degrees in biochemistry. I am currently Head of the Environmental Biochemistry Program at the Pacific Northwest Research Foundation, an independent, non-profit medical research facility, located in Seattle, Washington. I am an expert in the field of biochemistry and toxicology, particularly in relation to the effects of environmental chemicals on aquatic organisms and the etiology of cancer.

2. Other posts which I presently hold are an affiliate professorship in the University of Washington's Department of Environmental Health and a Research Professorship

in Chemistry at Seattle University. I am one of the founders of, and have been for many years the Editor-in-Chief of, the international journal Aquatic Toxicology. I have also served as a U.S. member of the Science Advisory Board of the International Joint Commission for the Great Lakes. A copy of my Curriculum Vitae is attached hereto as Exhibit A and is incorporated herein by reference.

3. Since 1967, my principal professional work has involved field and laboratory studies of the effects of environmental chemicals on aquatic organisms. In the last four years I have also been concerned with the role played by environmental chemicals in human cancer. I have specialized knowledge in the toxicology of chlorinated hydrocarbons, including the chemicals known as polychlorinated dibenzo-p-dioxins ("PCDDS" or "dioxins"). I have reviewed a document entitled Total Maximum Daily Loading (TMDL) to Limit Discharges of 2,3,7,8-TCDD (Dioxin) to the Columbia River Basin (the "TMDL") issued by USEPA on 25 February, 1991, [1] and EPA's Responses to Comments thereto. I have also reviewed portions of other documents including Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and -Dibenzofurans (CDDs and CDFs) and Integrated Risk Assessment for Dioxins and Furans from Chlorine Bleaching Pulp and Paper Mills, as well as scientific studies of the behavior of chlorinated hydrocarbons in the environment.

4. As I explain in this declaration, it is my professional opinion that the EPA's failures to consider the

complex toxic environment into which 2,3,7,8-TCDD is discharged and non-cancer effects on aquatic and wildlife species like bald eagles in establishing its TMDL are scientifically indefensible. The TMDL is predicated on scientific deficiencies that, if properly considered, would have indicated to EPA that its TMDL may well have adverse effects on bald eagles and other aquatically-dependent species.

5. The 2,3,7,8-TCDD is among the most potent animal carcinogens and one of the most potent reproductive toxins known [16]. The 2,3,7,8-TCDD is one of a family of related compounds which produce toxic responses mediated by an intracellular protein, called the Ah receptor [16]. When dioxin or one of the dioxin-like substances attaches to the Ah receptor in an animal cell, it causes particular regulatory and structural genes to be transcribed in the nucleus of the cell, inducing the production of several drug-metabolizing enzymes. One of the enzymes induced in large quantities is aryl hydrocarbon hydroxylase (AHH)[16]. The 2,3,7,8-TCDD produces effects, mediated by these biochemical processes, which include reproductive impairment [2, 3], cytogenetic changes [4, 5], cancer [5], immune system dysfunction [6] and wasting syndrome [7].

6. Further, the 2,3,7,8-TCDD is only one of a host of toxicologically significant compounds produced in the manufacture of chlorine-bleached pulp and paper waste, including polychlorinated dibenzofurans (PCDFs), chlorinated guaiacols, resin acids, and a host of other potentially toxic substances. It is well known that a number of these compounds act

interdependently, as do other organic compounds [8]. The TMDL nonetheless disregards the existence of these important chemical interactions in complex system, by reducing the number of toxic components under consideration to a single compound, 2,3,7,8-TCDD. This is apparently based on the unfounded assumption that laboratory-based exposure-toxicity relationships for 2,3,7,8-TCDD are valid under all circumstances, regardless of the interactive effects potentially associated with complex mixtures in pulp mill effluents and receiving waters. Even assuming that it were scientifically defensible to regulate 2,3,7,8-TCDD as if it occurred in isolation, an assumption which the scientific literature refutes [5, 9], EPA's failure to account for dioxin and other contaminants already existing in aquatic organisms and wildlife like bald eagles underestimates the risks to these organisms.

7. The lower Columbia River has been and continues to be subjected to tremendous environmental stress. Myriad chemical contaminants have been discharged into this river over time, and are presently occurring at levels causing chronic toxic effects to aquatically-dependent species residing there [10]. It is significant that fish, for example, are known to be contaminated with dioxin [1]. Elevated levels of DDE and PCBs have been found in bald eagle eggs [10], as have elevated concentrations of mercury [11]. Accumulations of PCBs and DDE have also been reported in mink, river otters, and harbor seals from the lower Columbia River [11]. PCB residues in mink exceeded concentrations shown to be associated with reproductive

impairment in these mammals [11].

8. Toxicologically, a chemically stressed system such as the lower Columbia River is less resilient in the face of an added environmental insult, such as that imposed by the addition of dioxin and myriad other toxic substances coexisting with it in pulp mill effluent. An organism exhibiting no toxicological response under laboratory conditions to a given concentration of contaminant, may, under these stressed conditions, be highly sensitive to the same concentration in a toxic milieu. Studies which I and others have conducted have established clear correlations between the accumulation of toxic chemicals and serious biological effects in aquatic organisms [12]. Just as rats fed fish contaminated with PCBs, dioxin and other organochlorines will exhibit increased reactivity to adverse events (e.g. behavioral changes) compared to rats fed uncontaminated diets [17], so is it likely that other life forms, such as fish-eating birds, will be affected by the chemical stresses occurring through their diets, particularly where the affected organisms already exhibit indications of toxic stress from accumulated chemicals.

9. There is absolutely no basis for EPA to dismiss the existence of other toxic compounds in its regulation of 2,3,7,8-TCDD with respect to its determination that there would be no adverse effects to consumers of contaminated aquatic organisms, such as bald eagles. Importantly, the EPA fails to explain how its "model system" relates to conditions pertaining in highly complex and diverse aquatic environments where stresses

from other potent toxic chemicals are already causing harm to fish and wildlife. In my opinion, the EPA's assumption that it can establish a "margin of safety" for aquatic species such as bald eagles based on the premise that the effects of 2,3,7,8-TCDD can be considered in isolation is not only fallacious, but would lead to an erroneous conclusion that the margin of safety is adequate. The fact is that accumulations of DDE and PCBs already present in bald eagles and other aquatically-dependent wildlife clearly suggests that there is no established margin of safety on which EPA could rely in estimating the risk to such species from additional loads of dioxin and other components of pulp and paper mill effluents. This is especially true in light of well-established effects on toxicity from interactions between different types of chemicals.

10. Further, there is absolutely no scientific basis for assuming, without further analysis, that cancer is the most sensitive or appropriate indicator of health effects in aquatically-dependent organisms. For example, there is no evidence to conclude that all species exhibit genotoxic and teratogenic responses to dioxin [5] at environmentally realistic concentrations and it is not clear that cancer is even the most sensitive endpoint in humans [5, 13]. Very few studies have been conducted that relate to the relative immunotoxic and reproductive effects of dioxin on the many diverse forms of aquatic life (including their highly sensitive early developmental stages [14]) inhabiting the Columbia River and environs. Thus, it should be understood that the use of cancer

as a measure of environmental impact, as emphasized by the EPA, is of dubious relevance to the protection of aquatic ecosystems because many forms of aquatic life do not manifest cancer on exposure to classic mammalian carcinogens which have been tested on laboratory animals [12]. The EPA's attempt to link findings from laboratory animal (e.g., rodent) studies to toxic effects of dioxin in a vast array of fish, birds and other wildlife is ludicrous and lacks scientific credibility.

11. As indicated, there are only a limited number of aquatic species that respond to toxic insults by getting cancer[14], and there is no persuasive evidence to indicate that dioxin is a carcinogen to bald eagles or the broad spectrum of fish and bird populations inhabiting the Columbia River. Accordingly, there is no justification to use cancer as a "biomarker" for chemical effects that can occur in many forms other than cancer (e.g. reproductive failure) in the wide spectrum of potentially impacted species, ranging from small invertebrates to bald eagles.

12. The inadequacy of dioxin carcinogenicity as a basis for assessing ecosystem effects is particularly inappropriate considering existing contamination in the lower Columbia River. This is a complex biological system which cannot be compared to the limited controlled conditions in a laboratory experiment. A laboratory determination indicating that certain concentrations of a compound in isolation produce cancer in a few varieties of laboratory animals is essentially meaningless if the additive/ synergistic/antagonistic effects of other compounds

are not considered. In order to demonstrate that its TMDL protects important resources such as bald eagles from reproductive effects, EPA would have to demonstrate that carcinogenicity is a legitimate marker for the effects of dioxin when combined in complex mixtures. However, no such evidence exists. The fact is that there is simply no foundation in toxicological science for assuming that dioxin's individual effects on diverse species of fish and birds is in any way the same as its effects in combination with the myriad chemicals present in the pulp effluent and already present in the aquatic environment.

13. Even if it could be shown that dioxin would behave in the real world environment of highly complex chemical interactions as it does in isolation in the laboratory, there is no reason to believe that the 0.013 parts per quadrillion standard would be adequate to protect aquatic organisms and wildlife--it is, in fact, little more than an assumption. Although the quantities are extremely small, for some sensitive life stages a NOAEL (no observable adverse effects level) has not been established. For example, a NOAEL for the chicken embryo, the most sensitive life stage for this avian species, has not been attained [15]. Thus, the concentration that results in essentially a lack of toxic effect in birds is uncertain, but nonetheless may occur at the very low concentrations that EPA proposes to allow to be discharged under its TMDL.

14. There are often vast differences in the ability of organisms to bioconcentrate toxic organic compounds, as well as

in their physiological responses to a given contaminant. Thus, it may be that charismatic species such as bald eagles--particularly in their sensitive, early developmental stages--will be affected, even at the low exposure concentrations proposed. Factoring in the additional problem of additive and synergistic effects of other toxic compounds in the Columbia River, and the paucity of information on relevant effects thresholds, it is highly questionable whether there is a credible scientific basis to justify the discharge of any dioxin to waters of the Columbia River.

CONCLUSION

15. In sum, the EPA failed to properly address the biological consequences of discharging additional dioxin to the Columbia River in light of existing and future discharges of other environmental contaminants affecting diverse species varying from small invertebrates to bald eagles; failed to account for toxicological stress imposed by existing contaminants on these species and the Columbia River environment; and erroneously used cancer as a biomarker to protect aquatic organisms and aquatically dependent species such as bald eagles.

I declare under penalty of perjury that the foregoing is true and correct to the best of my knowledge. Executed this 11th day of November, 1991, in Seattle, Washington.

Donald C. Malins.

DONALD C. MALINS

MALINS2.DEC

1. U.S. ENVIRONMENTAL PROTECTION AGENCY (USEPA). 1991. Total Maximum Daily Loading (TMDL) to Limit Discharges of 2,3,7,8-TCDD (Dioxin) to the Columbia River Basin. USEPA, Seattle, Washington.

2. SILBERGELD, E.K., et al. 1987. Experimental and Clinical Studies on the Reproductive Toxicology of 2,3,7,8-Tetrachlorodibenzo-p-dioxin. American Journal of Industrial Medicine; 11(2): 131-144.

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6. CHASTAIN, J.E., et al. 1985. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD)-induced immunotoxicity. International Journal of Immunopharmacology; 7(6): 849-856.

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11. U.S. FISH AND WILDLIFE SERVICE. 1990. Briefing on the Columbia River. Memorandum from Russell Peterson, Field Supervisor, U.S. Fish and Wildlife Service, Jan. 11, 1990. 6 pp.

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14. MALINS, D.C., AND G.K. OSTRANDER. 1991. Perspectives in Aquatic Toxicology. Annual Review of Pharmacology and Toxicology; 31: 371-399.

15. U.S. ENVIRONMENTAL PROTECTION AGENCY. 1990. Integrated Risk Assessment for Dioxins and Furans from Chlorine Bleaching in Pulp and Paper Mills. EPA 560/5-90-011, Pp. 16-17.

16. U.S. ENVIRONMENTAL PROTECTION AGENCY. 1989. Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated Dibenzo-p-Dioxins and -Dibenzofurans (CDDs and CDFs), Risk Assessment Forum, EPA/625/3-89/016, 8.

17. DALY, H.B. 1990. Reward Reductions Found More Aversive by Rats Fed Environmentally Contaminated Salmon. Neurotoxicology and Teratology; 13: 449-453.

EXHIBIT A

EXHIBIT A

DONALD C. MALINS, PhD, DSc
Director, Environmental Biochemistry Program
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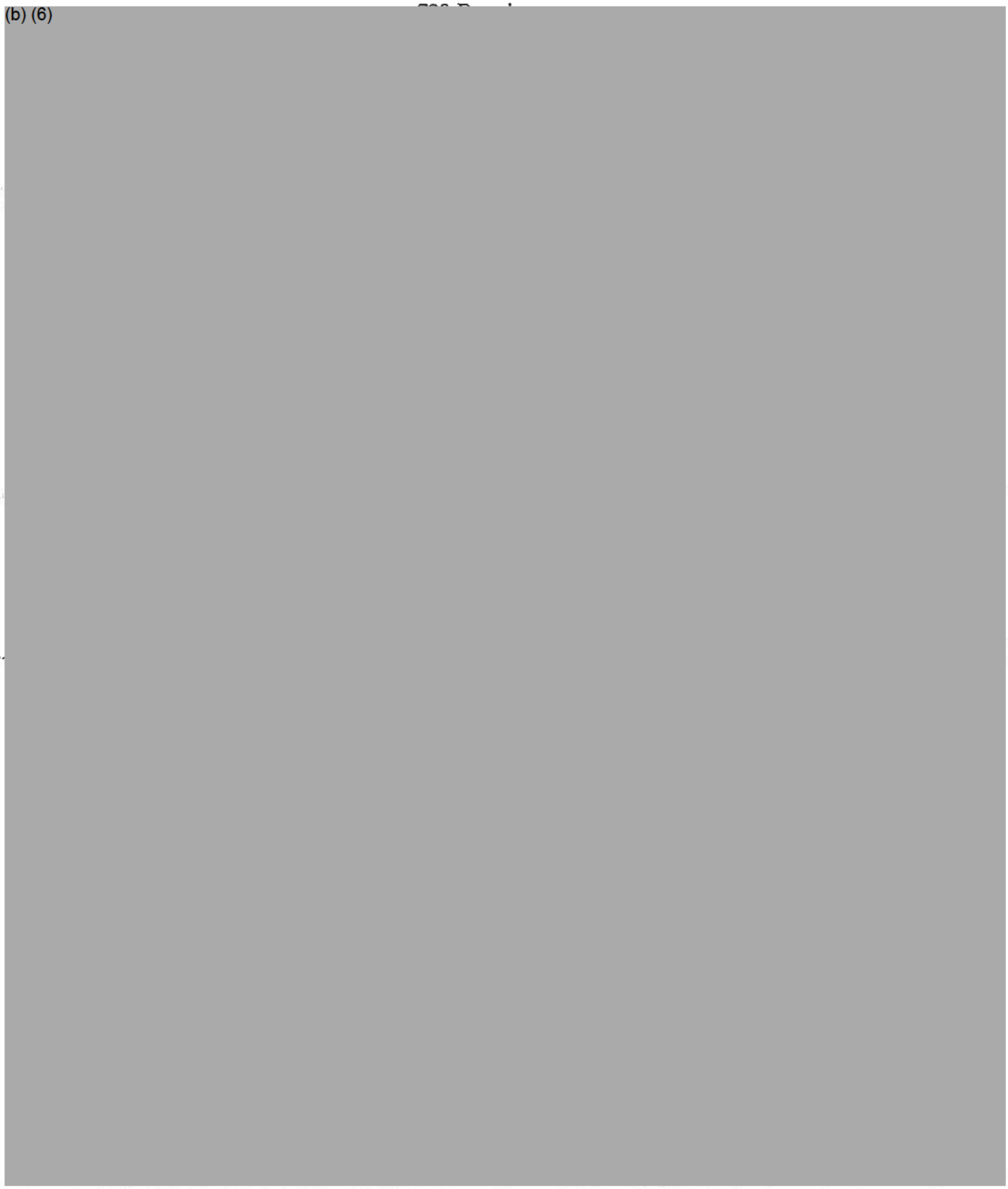

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